The Use of Topical Anesthesia in Evaluation & Treatment of Movement performance in neurological conditions

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Hong Kong 2006

SPASTICITY OR MUSCULAR HYPERTONIA: Definition
• Spasticity may be defined as a motor disorder characterized by a velocity dependent increase in the tonic stretch reflexes (muscle tone) with exaggerated tendon jerk, resulting from hyperexcitability of the stretch reflexes, as one component of the UMN syndrome. It is manifested by:
  1. Negative symptoms (performance deficit) a. Loss of strength  b. decrease dexterity.

Manifestations of Spasticity
• Positive symptoms (abnormal behavior):
  a. exaggerated tendon jerk (phasic reflexes), abnormal release functions (how to test?)
  b. Increased resistance to passive movement resulting in exaggerated tonic reflexes (how to test)
  c. Hyperactive flexion reflexes (muscle spasm)

Causes of Muscular Hypertonia
• Lesion to the extrapyramidal system at the CNS. (Cortical, capsular, brain stem, spinal cord; or basal ganglia as in cases of parkinson's disease). e.g. hemiplegia or para.
• Extrapyramidal system drives gamma & alpha MNs to balance the muscle tone. Lesion will cause imbalance that tip it toward hypertonia.
• +ve & -ve centers at CNS (balance & imbal.)

Classical concept of Hypertonia
• Based on hyperexcitability of selective MN pools e.g. Gamma, causing increased drive for muscle afferents & @-MN discharge & increased resistance to passive stretch. Based upon this hypertonia/rigidity may be categorized to a) gamma rigidity (treated by procaine or Phenol blocks of small diameter axons) & b) Alpha rigidity (survive gamma blockers, needs to target @MNs).

Classical concept/ Critique
• Based on decerebrate animal model (differs from human spasticity)
• Great variability in muscle hypertonia throughout the day depending on internal & external factors not related to gamma or alpha MNs. excitability.
• No hyperexcitability of selective gamma MNs was recorded with microneurography.
Current concept/ Spinal circuits
• Stretch reflex pathway (the reciprocal inhibition via Ia inhibitory interneurones).
• Inverse stretch (myotatic) reflex (autogenic inhibition of same muscle group by Ib inhibitory interneurones).
• The withdrawal (flexion) reflex: produce flexion of the stimulated limb & extension of the contralateral limb (what are the circuitries involved?) specify the inhibitory interneurones.

Current concept/ Neuronal Circuitries
• Group II inhibitory interneurones.
• Reshaw cell inhibition (recurrent inhibition) via inhibitory interneurones.
• Presynaptic inhibition via inhibitory interNs.
• Intersegmental reflexes: links between both lower limbs, upper limbs or U & LLs (e.g. withdrawal reflex) via propriospinal pathways and interneurones.

More on Neuronal circuits
• Supraspinal reflexes mediated by centers from brainstem, cerebellum, basal ganglia & other areas of the brain ON spinal neurons, relay via interneurones.
• Visceral (autonomic) reflexes that control thermal adjustment of vascular tone, gastrointestinal tract motility, urine storage & bladder emptying & sexual function.

Pathophysiology of Spasticity
• Reduced inhibitory mechanisms at the α-MNs due to disturbed interneurones functions:
  • Ia inhibitory interneurones (reciprocal inhibition)
  • Ib inhibitory interneurones (autogenic inhibition)
  • Group II inhibitory interneurones.
  • Cutaneous inhibitory input (interneurones)
  • Renshaw's cell inhibitory interneurones (collateral inhibition).

Disturbed interneurones causes DISINHIBITION
• The common denominator for all neuronal circuits is INTERNEURONES.
• Disordered interneurones causes reduced inhibitory functions of the interneurons that may result in hypertonia. The current concept for the causes of muscular hypertonia is based on the disturbed balance between the inhibitory & excitatory functions.

Interneurones are the Pathological sites
• Therefore evaluation should be directed to the functions of interneurones.
• Treatment should target restoring the balance between interneurons.
• Interneurons functions via inhibitory neurotransmitter.
Neurophysiological Changes in Muscular Hypertonia

- Most signal comes from outside the body are excitatory e.g. stretch, cutaneous, visual, auditory...etc.
- Most unwanted signal are inhibited at CNS.
- The excitatory signal from the supraspinal centers e.g. corticospinal tract, are modulated by peripheral spinal input (central & peripheral signals are compatible)

Noncompatibility between central & peripheral signal

- In muscular hypertonia, the central and peripheral signals are non-compatible resulting in over or under response

Evaluation Methods of Spasticity

- Clinical:
  - DTR, Tonic stretch reflex, Ashworth scale (& modified), Fugl-Meyer test, FIM, 10RRM
- Mechanical:
  - Pendulum test: half relaxation time when you let the leg fell during sitting
  - The tenometer.

Evaluation Methods/ Electrophysiological

- Phasic Reflexes: mechanical tendon reflexes, H-reflexes,
- Tonic Reflexes: Tonic vibration reflex, EMG/stretch velocity curve, clasp knife phenomena, lead pipe phenomena
- Multichannels EMG: recording the activity of different muscles, simultaneously, during specific tasks.
Evaluation Methods/
Mechanical

- Measurement of position, velocity & acceleration of movements during fast elbow flexion/extension movements.
- 10 RRM.
- Pendulum test; half relaxation time when you let the leg fell during sitting.
- Myometer.

Table 6: Correlation Coefficients (ICC) Between the Modified Ashworth Scale and Measures of the Stretch Reflexes in Two Test Sessions

<table>
<thead>
<tr>
<th>Stretch Reflex Measures</th>
<th>Session 1</th>
<th>Session 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Velocity sensitivity (EMGvel)</td>
<td>.52 (.05)</td>
<td>.57 (.042)</td>
</tr>
<tr>
<td>Amplitude, muscles at rest</td>
<td>.77 (.05)*</td>
<td>.67 (.017)</td>
</tr>
<tr>
<td>EMG</td>
<td>.80 (.003)*</td>
<td>.25 (.239)</td>
</tr>
<tr>
<td>Torque</td>
<td>.77 (.05)*</td>
<td>.74 (.007)*</td>
</tr>
<tr>
<td>Amplitude, muscles active</td>
<td>.86 (.232)</td>
<td>.21 (.209)</td>
</tr>
</tbody>
</table>

Values are reported as Spearman’s ICC and one-tailed p values derived from Student’s t test. Abbreviation: EMGvel, percentage of maximal EMG activity per degree. Statistically significant (p < .01) with Bonferroni adjustment applied to modify the critical level for the tests of significance.
Evaluation Methods/ Clinical

- **Ashworth scale**: to test functions
- **Fugl-Meyer test**.
- **Functional independent measure**.

Functional Consequence of Hypertonia

- Hypertonia will cause increased resistance to the already weak voluntary activities. This will result in reduced performance.
- Patient will be easily fatigued from making simple movements.
- Hemineglect: the patient is unaware of his paralyzed limb/s; therefore not using them in movements.

Rehabilitation Strategies

- Mild paralysis + severe hypertonia: reducing hypertonia will increase ease of movement and improve function.
- Mild paralysis + mild hypertonia =
- Severe paralysis + mild hypertonia =

### Movement parameters of the affected elbow and knee joints in spastic patient

<table>
<thead>
<tr>
<th></th>
<th>Elbow</th>
<th>Knee</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Excursion</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexion</td>
<td>107±6°</td>
<td>95±7°</td>
</tr>
<tr>
<td>Extension</td>
<td>177±8°</td>
<td>177±9°</td>
</tr>
<tr>
<td><strong>Velocity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexion</td>
<td>298±33%/sec</td>
<td>378±44%/sec</td>
</tr>
<tr>
<td>Extension</td>
<td>377±62%/sec</td>
<td>453±51%/sec</td>
</tr>
<tr>
<td><strong>Acceleration</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexion</td>
<td>55±10%/sec²</td>
<td>79±9%/sec²</td>
</tr>
<tr>
<td>Extension</td>
<td>62±9%/sec²</td>
<td>64±5%/sec²</td>
</tr>
</tbody>
</table>
Should we care about spasticity?

- Recent concepts of rehabilitation of stroke and upper motor neuron disorders: IIIsteps
- Strengthening exercises through the muscular hypertonia.
- Constrain induced therapy.
- Functional exercise therapy.

Rehabilitation of Spasticity with Topical Anesthesia.

- Studies on Normal subjects: I. Skin stimulation: cause H-reflex inhibition (short lived) II. Skin desensitization: cause H-facilitation & no ATR changes (i.e. possible reduced muscle spindle sensitivity), also caused reduced tonic vibration reflex (meaning reduced tonic excitability)

Results indicate reduced tonic and phasic components of the stretch reflex without reducing volitional control.

Concept: desensitization of the skin (mechanoreceptors) may reduce hyperactive stretch reflexes without affecting volitional activity. Application in patients with hypertonia will have treatment effect.
Fig. 1. Schematic diagram illustrating the sites of action of topical anesthesia on the H-reflex. The shaded area represents the region of the leg where anesthesia was applied.

Fig. 2. Typical effects of topical anesthesia applied to skin areas (A) and skin dermatomes (B) on the H-reflex. Each column represents the responses of a single individual before (control) and 30 min after application of the anesthetic. The solid line represents the mean of 10 consecutive responses.

Fig. 3. Heart rate (HR) and arterial pressure (ATP) changes in the post-anesthetic period. The time course of changes in HR and ATP after topical anesthesia is shown for both the control and the anesthetized group.

Fig. 4. Initial recovery time of the H-reflex, in all subjects, before and after topical anesthesia. The solid line represents the recovery time before anesthesia, while the dashed line indicates the recovery time after anesthesia.

Fig. 5. Ankle angle (degrees) and inversion during the post-anesthetic period. The time course of ankle angle and inversion changes is shown for both before and after topical anesthesia.
Topical Anesthetic-Induced Improvements in the Mobility of Patients with Muscular Hypertonicity: Preliminary Results

Mohamed A. Sobhany and Cedra J. De Luca

Summary: Application of a topical anesthetic on the skin of the upper and lower limbs of chronic stroke and brain injury patients resulted in significant improvements in mobility and reduction of spasticity. These improvements were observed within 45 minutes of application and persisted for at least 30 days post-anesthesia. Further studies are needed to confirm these findings.

Gait changes in stroke post Topical Anesthesia
Volitional upper extremity movements increased post topical Anesthesia

Application of Topical Anesthesia in Neurological conditions

- Application in stroke.
- Application in CP children.
- Application in Parkinson disease: No effect.
- Application in pathological clonus
- Application in essential tremor.
- Application in torticollis.

A DECREASE IN CLONUS AMPLITUDE BY TOPICAL ANESTHESIA

W. J. MILLIS, M.D. and R. S. TOZOS
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(Received for publication: June 3, 1965)

Summary

Topical anesthesia was studied for its effect on the clonus (a pathological clonus is a pathological clonus in which a patient can initiate clonus for approximately 10 seconds). The results showed that topical anesthesia significantly decreased the amplitude of clonus. A possible clinical application is suggested. Topical anesthesia may be a non-invasive method to reduce clonus amplitude.
Application in patients with CP

Jonathan (Supine, before TA)

Jonathan (sidelying, before TA)
Jonathan (Pronelying, before TA)

Jonathan (supine, 30 min. Post TA)

Jonathan (sidelying, 30 min. post TA)

Jonathan (prone, 30 min. Post TA)
Protocol for application of Topical Anesthesia to Patients with Neurological Disorders (Muscular hypertonia)

- Patient's Selection:
  - Muscular hypertonia of different level due to cortical, capsular, brain stem or spinal origin.
  - Hereditary cerebellar ataxia.
  - Essential tremor.
  - Cerebral Palsy
  - Pathologic clonus
  - Patients who might not respond to TA:
    - Parkinson's Disease.
    - Athetosis
    - Polynueropathy.
    - Peripheral nerve injuries.

- Testing Patients:
  - Patient must maintain a normal superficial skin sensation to cotton wool. Use cotton wool when you stimulate both upper limbs or lower limbs simultaneously and ask the patient if there is any difference in sensation between both sides. Patients with decreased superficial skin sensation should be excluded from the study.
  - Patients with reduced or loss of proprioceptive sensation might be excluded from the study also.
  - Patients who might be allergic to sprayed material or skin cream should be excluded.

- Topical Anesthesia to be used:
  - Lidocaine 5-10% oral spray of “Astra” pharmaceutical company is optimum. It is a prescription medication.
  - Lidocaine 5% cream. (also of Astra Pharmaceutical)
  - EMLA topical anesthetic (also of Astra Pharmaceutical)
  - Benzocaine 20%. This is off shelves and can be found in sunburn application (sprays). They are of different brands.

- How to Apply topical Anesthesia?:
  - TA should applied (sprayed or spread on all skin areas of the lower limbs EXCEPT the anterior tibial skin area (from the anterior border of the tibia to the line drawn from the lateral malleolus to the head of the fibula. This selected skin areas/dermatome may be used.

Jonathan (30 min post TA): passive hip abduction
• TA should be applied on all skin areas of the upper limb EXCEPT the posterior forearm (skin area between the elbow, wrist and radius & ulnar boundaries). This might be useful for patients with flexion synergies.
• Apply enough TA to the skin, massage the skin well for few seconds until you are sure from skin absorption !!! Patient to take a rest for 30 min. and then test patient for reduced muscular hypertonia and increased volitional movements.

• Always apply TA 30 min. before the therapy session. Effect might last for over 6 hours (or even longer in tremor patients).
• Apply TA 3X/wk for 2-3 months (if needed). The skin will show adaptation to the type of topical anesthetic after 2 months. Change the type of anesthetic and continue if needed.
• Patient is requested not to wash the TA or to bring arm/hand toward

Side effect of TA application:
• Some patients might feel sleepy after application of TA due to muscle relaxation.
• Patient might feel the sticking of the skin after TA application.

Use of Topical Anesthesia with Other Treatment procedures
• Topical Anesthesia + Constrain induced therapy.
• Topical anesthesia + Task-oriented therapy/exercises.
• Topical Anesthesia + soft tissue stretching techniques.
• Topical anesthesia + body weight support training.
• Topical anesthesia + NDT

The use of topical anesthesia in Essential tremor
• Pozos et. al 1992.
• Our study.
• The video

Conclusions
• Topical Anesthesia may be useful in improving movement performance in patients with stroke, pathologic clonus, essential tremor and CP, by reducing muscular hypertonia & augmenting volitional activity.
• Patient's selection is central for the effectiveness of the technique.
Bibliography


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